In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
Case No. 10-077V
Filed: May 19, 2015

* *	**PUBLISHED**
D.S., *	
*	Special Master Dorsey
Petitioner, *	
v. *	Entitlement; Human papillomavirus (HPV) vaccine; Gardasil; Guillain-Barré syndrome (GBS); Miller-Fisher Variant; Significant Aggravation.
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SECRETARY OF HEALTH *	
AND HUMAN SERVICES, *	
*	
Respondent. *	
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* *	

Thomas P. Gallagher, Somers Point, NJ, for petitioner.
Darryl R. Wishard, U.S. Department of Justice, Washington, D.C., for respondent.

RULING ON ENTITLEMENT[1]

I. Introduction

On February 12, 2010, D.S. ("petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("the Program" or "Vaccine Act"),[2] alleging that she suffered from the Miller-Fisher variant of Guillain-Barré syndrome ("GBS") in April 2007, as a result of receiving the first dose of the human papillomavirus ("HPV") vaccine ("Gardasil") on February 21, 2007. Petition at 2. Respondent recommended against awarding compensation to petitioner, stating that petitioner had not presented preponderant evidence that

1 When this decision was originally issued, the parties were notified that the decision would be posted in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, § 205, 116 Stat. 2899, 2913 (codified as amended at 44 U.S.C. § 3501 note (2006)). Petitioner was also notified that she could seek redaction pursuant to § 300aa-12(d)(4)(B); Vaccine Rule 18(b). Petitioner made a timely request for redaction and this decision is being reissued in accordance with the request for redaction.

2 The Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C.§§ 300aa-10 et seq. (2006). Hereafter, individual section references will be to 42 U.S.C. 300aa.

the HPV vaccination caused her injuries. See Respondent's Rule 4 Report ("Resp't Report"), filed March 11, 2011, at 1, 14.

The parties submitted expert reports in support of their respective positions. Petitioner filed several expert reports from David Axelrod, M.D., an immunologist. Petitioner's Exhibits ("Pet. Ex.") 23, 40, 41, 43. Petitioner also filed expert reports from one of her treating neurologists, Steven H. Schechter, M.D. Pet. Exs. 56, 57. Respondent filed reports from Thomas Leist, M.D., a neurologist and neuroimmunologist. Respondent's Exhibits ("Resp't Ex.") A, G.

A hearing was held on August 20, 2014, during which the parties' experts testified. Petitioner filed a post-hearing brief on December 16, 2014. Respondent filed her post-hearing brief on December 23, 2014. The matter is now ripe for adjudication.

After a review of the entire record, the undersigned finds that petitioner has provided preponderant evidence that she developed the Miller-Fisher variant of GBS after her February 21, 2007 Gardasil vaccination. The undersigned also finds that petitioner has provided preponderant evidence that the Gardasil vaccine caused her to develop the Miller-Fisher variant of GBS, which satisfies her burden of proof under Althen v. Sec'y of Health & Human Servs., 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, petitioner is entitled to compensation.

II. Issues to Be Decided[3]

In their joint prehearing submission filed on September 18, 2014, the parties presented several issues in dispute. To decide the case, the parties request that the undersigned first determine whether petitioner has presented preponderant evidence that she suffered from an onset of the Miller-Fisher variant of GBS or an autoimmune[4] demyelinating[5] disorder after her receipt of the HPV vaccine on February 21, 2007. See Amended Joint Submission at 2, filed Sept. 18, 2014.

Second, the parties request that the undersigned determine whether petitioner has presented preponderant evidence that she suffered from an identifiable, underlying medical condition before February 21, 2007. Id.

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[3] While the parties have requested that the undersigned determine whether petitioner suffered from an "onset of the Miller-Fisher variant of GBS," an "autoimmune demyelinating disorder" or an "identifiable, underlying medical condition," the undersigned did not limit her analysis to just these conditions, but also considered whether petitioner could recover for any illness or injury. See § 300aa-11(c)(1)(C)(ii)(I).

[4] "Autoimmune" is characterized "by a specific humoral or cell-mediated immune response against constituents of the body's own tissues (self antigens or autoantigens). Dorland's Illustrated Medical Dictionary ("Dorland's") 181 (32d ed. 2012).

[5] Demyelination is the "destruction, removal or loss of the myelin sheath of a nerve or nerves." Dorland's at 486.

Third, the parties ask that the undersigned determine whether petitioner has presented preponderant evidence for each factor under Althen, 418 F.3d at 1278, to show that the HPV vaccine she received on February 21, 2007 was, more likely than not, a substantial factor in causing the onset of her symptoms on April 3, 2007, as alleged by petitioner as the Miller-Fisher variant of GBS or an autoimmune demyelinating disorder. Id.

Finally, the parties request that the undersigned decide under Loving v. Sec'y of Health & Human Servs., 86 Fed. Cl. 135, 144 (2009); see also W.C. v. Sec'y of Health & Human Servs., 704 F.3d 1352, 1357 (Fed. Cir. 2013) (holding that "the Loving case provides the correct framework for evaluating off-table significant aggravation claims"), whether petitioner has presented preponderant evidence that the HPV vaccine significantly aggravated an underlying medical condition as of April 3, 2007. Id.

Thus, the parties have asked the undersigned to determine the nature of petitioner's injury and to determine whether the Gardasil vaccine caused or significantly aggravated that injury.

III. Procedural Background

Petitioner filed her petition for compensation on February 12, 2010. Petition (ECF No. 1). Over the next year, petitioner filed the relevant medical records and certified completion of the record on January 28, 2011. ECF No. 30. On March 11, 2011, respondent filed her Rule 4(c) Report "(Resp't Rept.") stating that this case was not appropriate for compensation because petitioner had not presented sufficient evidence of causation under all three prongs of Althen, 418 F.3d at 1278. Resp't Rept. at 1, 19 (ECF No. 31). Respondent argued that in addition to presenting insufficient evidence on causation, petitioner's diagnosis of GBS or the Miller-Fisher variant was highly unlikely. Id. at 15-16.

Thereafter, the case proceeded on a dual litigation/settlement track where the parties proceeded with filing expert reports, while at the same time attempting to informally resolve the case. On November 29, 2011, petitioner filed an expert report from David Axelrod, M.D., and supporting medical literature. ECF No. 39. On February 10, 2012, respondent filed a responsive expert report from Thomas P. Leist, MD, PhD, along with his curriculum vitae and the medical literature references from Dr. Leist's expert report. ECF No. 47. On April 12, 2012, and May 8, 2012, petitioner filed additional supplemental expert reports from Dr. Axelrod. ECF Nos. 49, 52. The special master previously responsible for this case set a hearing for September 14, 2012. ECF No. 58.

Respondent filed another responsive expert report from Dr. Leist on August 13, 2012. Resp't Ex. G (ECF No. 66). A few days thereafter, petitioner's attorney filed a motion to withdraw as counsel due to his termination by petitioner. ECF No. 67. New counsel for petitioner filed a consent motion to substitute as counsel on August 21, 2012. The motion was granted. ECF No. 69. As a result of petitioner's retention of new counsel, the hearing previously set for September 14, 2012, was continued, and new deadlines were set for the filing of additional expert reports. ECF No. 70. The parties worked to resolve petitioner's application for an award of interim attorneys' fees and costs to her prior counsel, and a decision on interim

fees was entered on November 16, 2012. ECF No. 78. This case was then reassigned to the undersigned special master on January 14, 2013.

The undersigned special master conducted a status conference with the parties on March 12, 2013. The parties were ordered to discuss whether an informal resolution of the case was appropriate. Petitioner's pending motion to file an additional expert report was also granted. ECF No. 85.

On May 1, 2013, and May 23, 2013, petitioner filed expert reports from her treating neurologist, Dr. Steven Schechter. ECF No. 89, 92. Petitioner also filed her own affidavit in support of her claim on June 6, 2013. ECF No. 93. At a status conference held on June 13, 2013, respondent's counsel indicated that respondent was not interested in pursuing informal resolution of this case and requested that the case be set for hearing. Deadlines were set for the filing of updated medical records and any additional expert reports. ECF No. 94. A hearing was set for March 19-20, 2014. ECF No. 95.

After filing several motions for enlargements of time, petitioner filed a supplemental expert report from Dr. Schechter on October 31, 2013. ECF No. 101. On December 9, 2013, respondent filed a supplemental report from Dr. Leist addressing the most recently filed medical records and Dr. Schechter's most recent report. ECF No. 102.

On March 14, 2014, due to an urgent issue, staff from petitioner's counsel's office contacted the court to request that the March 19-20, 2014 hearing dates be continued. Respondent had no objection. ECF No. 114. The hearing was rescheduled for August 20, 2014, and proceeded as scheduled. Petitioner testified on her own behalf, along with Dr. Axelrod and Dr. Schechter. During the hearing, petitioner's counsel presented a report from Dr. Axelrod (dated August 2012) that had not been filed into the record. Because respondent's counsel and respondent's expert, Dr. Leist, had not had the opportunity to review the report or the literature cited in that report prior to the hearing, the undersigned allowed respondent the opportunity to file a supplemental expert report after the hearing. After the hearing concluded, petitioner was ordered to file Dr. Axelrod's report and a deadline was set for respondent to file a responsive expert report.

A post-hearing status conference was held on August 25, 2014. At the status conference, petitioner's counsel stated that on the basis of the testimony heard at the hearing, petitioner was now interested in pursuing a significant aggravation claim. The undersigned ordered the parties to file an amended joint submission listing the significant aggravation claim as an issue in dispute. The parties were also encouraged to revisit settlement discussions and a deadline of September 24, 2014, was set for the petitioner to file a status report indicating that a demand had been sent to respondent. ECF No. 118. The parties filed the amended joint submission on September 18, 2014. ECF No. 124. Petitioner also filed a status report on September 22, 2014, stating that a demand had been sent to respondent. ECF No. 125.

On October 9, 2014, respondent filed a status report stating that respondent would not be filing a supplemental expert report from Dr. Leist, as respondent believed that Dr. Leist's prior reports and hearing testimony addressed the issues raised in Dr. Axelrod's August 2012 report.

Respondent also stated that she would file a brief on the significant aggravation claim only if petitioner elected to pursue that issue and filed a brief. ECF No. 127.

On December 16, 2014, petitioner filed a post-hearing brief and a supplemental expert report from Dr. Axelrod in support of petitioner's significant aggravation claim. ECF Nos. 132-33. Respondent filed her post-hearing brief on December 23, 2014. ECF No. 134.

IV. Factual Background and Medical History[6]

A. Petitioner's Pre-Vaccination Medical History

Petitioner was born on [redacted], 1956. Amended Joint Submission ("Amended. Jt. Sub.") at 1, filed Sept. 18, 2014. A review of petitioner's medical records that pre-date her HPV vaccination demonstrate [redacted] that petitioner had a lengthy and complicated pre-vaccination medical history. Id. The records include references to possible [redacted] musculoskeletal, [redacted] arthralgia, Lyme disease, mitral valve prolapse, [redacted], myalgias, right meniscus tear, fibromyalgia, rheumatoid arthritis, chronic fatigue, right hearing loss, [redacted] See Amended Jt. Sub. at 1; Pet. Ex. 11 at 4-9, 54; Pet. Ex. 17 at 7-24, 78-105, 195-218.

On May 3, 2001, petitioner presented to William M. Leuchter, M.D. (neurologist), with complaints of acute hearing loss and migraine headaches. Pet. Ex. 17 at 209-10. Also in May 2001, petitioner presented to A. Martin Lerner, M.D. (an infectious disease specialist) with symptoms of right facial numbness, blurry eyesight, decreased hearing, and diagnoses of rheumatoid arthritis and fibromyalgia. Id. at 206-08. Petitioner told Dr. Lerner that she had Meniere disease[7] with symptoms of bilateral pressure [redacted] Id. at 209.

In a history provided by petitioner on June 12, 2001, and taken by Jeffrey D. Band, M.D., another infectious disease physician, petitioner noted that she had been in good health prior to 1993, but she thereafter developed fatigue, arthralgias, myalgias, and intermittent swelling of her glands and lymph nodes. Pet. Ex. 17 at 203-04. Petitioner explained that in more recent years, she developed right hearing loss and right visual changes. Id. Dr. Band noted in his report that he found little evidence of Lyme disease as the serum test did not meet criteria for positivity. Id. at 203. On June 22, 2001, petitioner had an abnormal visual evoked potential test showing mild optic nerve dysfunction bilaterally, but more severe on the left. Id. at 57. On August 16, 2001,

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[6] This Factual Background and Medical History section contains a review only of the most relevant facts, although the undersigned has considered the record as a whole in reaching her decision. A more detailed recitation of the facts may be found in respondent's Rule 4(c) report and in the parties' respective post-hearing briefs.

[7] "Meniere disease" is defined as "hearing loss, tinnitus, and vertigo resulting from non-suppurative disease of the labyrinth with edema." Dorland's at 539.

during her annual exam, it was noted by petitioner's gynecologist that petitioner had hearing loss and facial nerve dysfunction, as well as difficulty emptying her bladder. Pet. Ex. 16 at 7.

An MRI of petitioner's brain and orbits was conducted on October 28, 2002, because of her continuing complaints of right eye pain. Pet. Ex. 17 at 97. The results were normal. On April 1, 2003, petitioner reported severe headaches and right hearing loss. Pet. Ex. 11 at 5. An MRA of petitioner's head on October 15, 2003, was negative for abnormalities. Pet. Ex. 17 at 96.

On January 7, 2004, petitioner reported that she was [redacted] continuing her antibiotic treatment for Lyme disease and [redacted] Pet. Ex. 11 at 4. A Lyme test on March 15, 2004, again did not meet criteria for seropositivity. Pet. Ex. 17 at 59.

On February 24, 2004, petitioner saw Robert W. Ike, M.D., a rheumatologist, who had regularly treated her for years. Pet. Ex. 17 at 211-13. Dr. Ike noted that he had not seen petitioner in nearly three years [redacted] Id. at 211. At that time, petitioner complained of ongoing pain, stiffness and fatigue despite antibiotic treatment for Lyme disease. On exam, Dr. Ike found no evidence of ongoing synovitis, but did note that petitioner seemed to have experienced hearing loss. Id. at 212. In summarizing his findings, Dr. Ike stated that petitioner had been "a diagnostic challenge to the many physicians who had seen her over the years with no satisfactory explanations or treatments for her various symptoms, which ranged from annoying to debilitating." Id.

On October 9, 2006, petitioner treated at the Michigan Ear Institute for complaints of worsening hearing loss, lack of balance with several falls in the past year, facial paresis and numbness, and a history of Lyme disease. Pet. Ex. 19 at 6-7, 17-18. The exam showed slight facial weakness on her right side. Id. Petitioner's physical exam was otherwise normal with the exception of some slight instability noted on her balance tests. An exam on October 30, 2006, at the Michigan Ear Institute noted that petitioner had a healthy appearance, that she was "alert and oriented," and that her facial function was normal. Id. at 16. Some degree of hearing loss in both ears was noted. Id. On November 20, 2006, petitioner was seen by her gynecologist, who noted that petitioner was "doing well" and that she had "no new medical problems." Pet. Ex. 11 at 3. [redacted]

B. February 21, 2007 HPV Vaccination and Subsequent Medical History

[redacted] Petitioner testified during the hearing that her doctor recommended that she received the HPV vaccine because it was "protective for all women." Transcript ("Tr.") at 9. She received one dose of the HPV vaccine at her next gynecological visit on February 21, 2007, at age 50 years. Pet. Ex. 11 at 2. There were no reported immediate side effects.

Petitioner testified that the onset of her symptoms began on April 1, 2007 (39 days after vaccination), when she started having a twitching sensation in her face and experienced extreme

fatigue. Tr. at 9. She explained that the symptoms got "progressively worse" over the next couple of days and she felt like she was "being hit with a ton of bricks." Id. at 9-10.

On April 4, 2007 (42 days after vaccination), petitioner presented to the William Beaumont Hospital ("WBH") ER with complaints that began the day before of a "frozen face," difficulty eating, swallowing, talking, and a headache, and a history of hearing loss, Lyme disease, and migraine. Pet. Ex. 10 at 12-14. On examination, petitioner had facial droop, implicating the right seventh cranial nerve. Id. at 19. The results of lab testing were normal, and petitioner was discharged that same day from the ER with a diagnosis of Bell's palsy. She was instructed to follow-up with Dr. Lerner, her infectious disease physician, who was consulted during the ER visit. Id.

Petitioner returned to the ER later that same day on April 4, 2007, with complaints of facial numbness and paralysis, an inability to swallow, and slurred speech, which started the prior day. She was admitted to the hospital at this time. Pet. Ex. 10 at 13-14, 16-17. The impression at admission was cranial nerve palsies. Id. at 17, 54, 57, 82. A blood test showed a positive Lyme IgG/IgM screening. Id. at 159. A lumbar puncture was normal for red cells, white cells, protein, Lyme PCR, arboviruses, and acetylcholinesterase. Id. at 160-65. Antiganglioside antibodies, including GQ1b, were negative. Pet. Ex. 11 at 20-21.

The Patient Discharge Summary notes (which summarized each date of petitioner's hospitalization), stated for the April 5, 2007 hospitalization date, that petitioner was wearing a hearing aid on both sides, and that she had a history of rheumatoid arthritis, Lyme disease, deafness to her right ear, knee surgery, [redacted] Pet. Ex. 10 at 223-24. In the physician admission history, it was noted that petitioner's symptoms started on April 4, 2007, and that she had a history of migraines [redacted] Pet. Ex. 10 at 54. Facial numbness, very diffuse facial/cranial paresthesia, and dysphasia were all noted. Pet. Ex. 10 at 56.

The progress notes from WBH hospital dated April 5, 2007, state that petitioner [redacted] she had difficulty with her vision, smiling, speaking, and hearing afterwards. Pet. Ex. 10 at 58. Dr. Lerner examined petitioner and noted that he had not seen petitioner in three years. Id. He stated that a full neurologic examination of petitioner showed no paralysis, no Babinski, and no cranial nerve signs. Dr. Lerner also found no firm neurologic abnormalities. He noted petitioner's history of migraine headaches and seronegative Lyme disease. Id. at 58.

Saraswati A. Muttal, M.D., a neurologist consulted to evaluate petitioner for cranial nerve palsies, noted that petitioner reported an onset of ear pain and inability to close her eyes followed by loss of taste the day before coming to the hospital (April 3, 2007). Pet. Ex. 10 at 327. Petitioner's vital signs and physical exam were normal, with no motor weakness or sensory changes in her extremities, no ataxia, and normal tandem gait. Her deep tendon reflexes were normal at 2/4 bilaterally. Dr. Muttal mentioned that she was awaiting the results of an MRI of the brain. Id. Petitioner saw Dr. Muttal several more times in consultations during her hospitalization. Dr. Muttal commented on April 11, 2007 that petitioner's bilateral facial weakness was most likely due to [redacted] or Lyme disease. She stated that petitioner

wanted an opinion from Dr. Leuchter or another neurologist. Pet. Ex. 10 at 72-73. Lori A. Stec, M.D., an ophthalmologist, also saw petitioner on April 5, 2007, and diagnosed her with early exposure keratopathy secondary to Bell's palsy. Pet. Ex. 10 at 85.

A brain MRI, with and without contrast, was performed on April 5, 2007, and the results were compared to previous studies conducted in October 2002 and October 2003. Pet. Ex. 10 at 140-41. The results of the April 2007 MRI were read as within normal limits, [redacted] The results of petitioner's brain MRA and MRV[8] (dated April 5, 2007) were unremarkable. Id. A cervical spine MRI (with and without contrast), conducted on the same date showed degenerative disc disease but no impinging lesions. Pet. Ex. 10 at 141. On April 10, 2007, electromyogram and nerve conduction studies ("EMG/NCV") showed evidence of bilateral seventh cranial nerve neuropathy with no voluntary function of muscle groups supplied by the seventh cranial nerve. It was recommended that GBS be ruled out. Pet. Ex. 10 at 109. In a consultation on April 11, 2007, with Raina M. Ernstoff, M.D., a neurologist, differential diagnoses of myasthenia gravis, Miller-Fisher syndrome, and multiple sclerosis were entertained. Pet. Ex. 10 at 155-56. An MRI of the internal auditory canal on April 14, 2007, was read as showing enhancement of the distal intracanalicular portions of the auditory canals and of the descending facial nerves. Id. Petitioner was started on long-term IV antibiotics. Pet. Ex. 10 at 347.

On April 12, 2007, petitioner was seen by Myron Laban, M.D., a physiatrist, who noted petitioner's bilateral facial paralysis with improving function on the right but absent function on the left. Pet. Ex. 4 at 339-41. Dr. Laban stated that there was no evidence of ataxia and petitioner's proprioception was intact. Petitioner had no lower extremity weakness – her deep tendon reflexes were not tested. Dr. Laban noted that petitioner's EMG study was suggestive of bilateral neuropathy of the facial nerve. Dr. Laban felt that petitioner had a "classic presentation of Guillain-Barré [] syndrome of acute bilateral facial paresis." Id. at 340.

Petitioner was discharged from WBH on April 16, 2007, by Dr. Lerner. Pet. Ext. 10 at 347. In the discharge summary, Dr. Lerner noted that petitioner was admitted to the hospital with a sudden inability to speak, close her eyelids, move her face, or swallow. He also noted that petitioner's "abnormalities were real and neurologic," although the CT and MRI scans of her head were normal. Id. Because petitioner's Lyme serology was positive, a diagnosis of cerebral Lyme disease was made, although it was noted that a number of diagnostic studies were also being conducted. Id. Dr. Lerner noted that petitioner was on intravenous antibiotics and that long-term administration of the antibiotics was contemplated. Id.

Petitioner underwent EMG and NCV studies on April 19, 2007, which showed dysfunction in her bilateral fifth and seventh cranial nerves. Pet. Ex. 17 at 75-76. The study was interpreted by Kirsten Gruis, M.D., a neurologist, who stated that petitioner's normal cerebral spinal fluid ("CSF") analysis and CSF Lyme Western blot results "argue against an infectious process or demyelinating polyneuropathy variant." Pet. Ex. 17 at 75-76. Dr. Gruis noted that idiopathic cranial neuropathies or autoimmune collagen-vascular disease could explain the multiple cranial mononeuropathies. Id.

[8] Magnetic Resonance Venography

In a separate office note, dated April 19, 2007, Dr. Gruis recounted petitioner's medical history including her longstanding history of rheumatologic symptoms, joint swelling, intermittent sharp pain in her right eye since the late 1990s, sensory neural hearing loss since 2001, and chronic headaches. Pet. Ex. 17 at 156-57. Dr. Gruis noted that earlier on April 1, 2007 (the day of onset of petitioner's symptoms), petitioner was at a garden party and ate salmon and spinach and began developing neurologic symptoms later in the day. Petitioner told Dr. Gruis that her symptoms began with a sensation on the left side of her face of twitching without any associated numbness or tingling. Id. When she woke the next morning, petitioner stated that the right side of her face was motionless. Id. Over the course of the next day, petitioner stated that she began to develop facial paralysis on the left side of her face. Dr. Gruis noted that petitioner was taken to WBH where she was thought to have bilateral facial nerve palsy; [redacted] Later, nerve conduction studies performed on petitioner's facial nerves demonstrated that petitioner more likely had facial nerve palsy. Id. at 156. On examination, Dr. Gruis noted that petitioner had some subjective weakness on her right eye closure, but there was no Bell's phenomenon noted. Id. Petitioner was able to slightly raise her left eyebrow but was unable to close her left eye. Id. In her assessment, Dr. Gruis stated that the CSF findings made "infectious etiologies as well as acute inflammatory demyelinating polyradiculoneuropathy (AIDP, Guillain-Barré syndrome) very unlikely causes of [petitioner's] symptoms." Id. at 159. Dr. Gruis noted that the results of petitioner's "limb EMG done at her local hospital have normal F-wave responses arguing against Guillain-Barré syndrome as well." Id. Dr. Gruis felt that the most likely etiology of petitioner's entire symptom complex of bilateral cranial nerve involvement likely represented a systemic autoimmune disorder or collagen vascular disease. Id.

On April 23, 2007, petitioner presented to Dr. Lerner for a consultation. Upon examination, Dr. Lerner commented that petitioner was "remarkably better." Pet. Ex. 21 at 9. He noted that petitioner's enunciation was clear and that she could close her right eye. Id. Dr. Lerner also noted that he saw very little weakness on the right side of petitioner's face, and no weakness in her arms and legs, and noted that her neurologic exam was otherwise normal. Dr. Lerner noted petitioner's [redacted] diagnosis of "midbrain Lyme disease" and his impression stated: [redacted] Id.

Dr. Ike (a rheumatologist) saw petitioner on April 30, 2007, noting that it was petitioner's first visit to him in two years. Pet. Ex. 17 at 136-38. Dr. Ike noted that petitioner had a consultation and examination with Dr. Gruis who noted that petitioner did have facial nerve palsy but that he did not see evidence of Lyme disease. Id. at 137. Dr. Ike stated that petitioner "seems to have a slowly resolving acute collection of cranial nerve palsies. The evaluation to date does not point towards a specific explaining diagnosis. Calling this 'autoimmune' does not really shed any light on the cause of the situation, but supports a way forth with suppressive corticosteroids . . . In the ten years that she has been coming here as a patient, I have failed to turn up anything to support a definable rheumatic disease process." Id. at 137-38.

On May 1, 2007, petitioner was seen by Justin C. Riutta, M.D., a physiatrist, who noted that petitioner's previous evaluations at the University of Michigan did not support a diagnosis of Lyme disease. Pet. Ex. 2 at 19-20. Dr. Riutta's impression was bilateral cranial nerve VII palsy.

Id. On May 7, 2007, petitioner consulted Dr. Ernstoff (neurologist) who performed a complete neurologic examination. Pet. Ex. 14 at 6. Dr. Ernstoff noted that petitioner had a normal exam except for incomplete bilateral corneal reflexes with incomplete blink, bilateral Bell's phenomena, and weakness of her right orb. Id. at 6-7. Dr. Ernstoff commented that bilateral facial nerve palsy can be seen in viral [redacted] infections. Pet. Ex. 14 at 6-7.

Petitioner next presented to Dr. Steven Schechter for neurologic evaluation on May 14, 2007. Pet. Ex. 3 at 24. Petitioner told Dr. Schechter that she believed she developed Lyme disease while on a cruise in Great Britain at age 36, and that she was diagnosed eight years later. Id. Petitioner stated that she initially thought her symptoms were a reaction to a diphtheria vaccination. Id. Petitioner reported that her current symptoms included hearing loss, stiff neck, an inability to drive, facial numbness, and headaches. Id. On exam, she had bilateral facial weakness and decreased facial sensation. Id. at 25. Petitioner's extremity strength, sensation and deep tendon reflexes were all normal. Id. Dr. Schechter opined that the facial diplegia could be post viral or related to her diagnosis of Lyme disease. Id.

On May 23, 2007, petitioner consulted with Dr. Sandro K. Cinti, an infectious disease physician. Pet. Ex. 17 at 175-78. Dr. Cinti noted that he had previously consulted with petitioner in 2001 because of a concern petitioner had about Lyme disease. Id. at 175. Dr. Cinti noted that petitioner had only one band on an IgM Western blot in 2001, and that these test results did not fit the CDC criteria for Lyme disease. Id. Dr. Cinti noted that "an extensive workup, even recently, showed no signs of autoimmune disease." Id. at 175. In his impression, Dr. Cinti stated that it was "still not clear to me that this is Lyme disease." Id. at 176. He suggested that petitioner seek additional evaluations to help make the diagnosis of neuro-Lyme disease. Id.

Petitioner underwent a brain MRI and an MRI of her internal auditory canal (with and without contrast) on May 30, 2007, which noted an "enhancement [of the] bilateral facial nerve at labyrinthine segment and geniculate ganglia portion. These findings are stable." Pet. Ex. 17 at 71-72. The impression stated "stable appearances of enhancement of bilateral facial nerves. . . ." Id. The radiologist noted that based upon a review of the medical literature, "only 10% of Bell's palsy presented bilaterally. Bilateral Bell's palsy can be seen, not limited to, in patients with Melkenson Rosenthal syndrome, Mobius syndrome, [Guillain]-Barré, Myasthenia gravis." Id.

Petitioner returned to Dr. Gruis on June 14, 2007. Dr. Gruis took into consideration petitioner's previous visit, where there was not enough objective evidence to support a Lyme disease diagnosis. Pet. Ex. 17 at 151. Due to petitioner's continued concerns, Dr. Gruis stated that petitioner was being referred to Dr. Nadelman, a Lyme disease specialist in New York, who would conduct a further evaluation for neuro-Lyme disease. Id. Dr. Gruis stated in her summary that she suspected that petitioner did not have neuro-Lyme disease based on her CSF Lyme PCR, as well as a negative IgM Lyme lab result. Id. Dr. Gruis also stated that petitioner "does not have a Miller Fisher Variant of Guillain-Barré Syndrome on EMG or on CSF labs. The most likely explanation of her bilateral facial nerve palsies is a viral neuritis from possibly varicella-zoster or another virus versus a mixed connective tissue disease." Id.

Petitioner returned for a follow-up consultation with Dr. Riutta on June 15, 2007. Pet. Ex. 2 at 17. Dr. Riutta noted that petitioner had recently been seen at the University of Michigan and diagnosed with idiopathic bilateral Bell's palsy with involvement of the seventh cranial nerve only. Id. Dr. Riutta also noted that Lyme disease serologies had been negative. Pet. Ex. 2 at 17-18. She recommended a follow-up for repeat electrodiagnostic studies if needed. Id. at 18.

Petitioner underwent another brain MRI on June 19, 2007. Pet. Ex. 17 at 70. The MRI showed previously noted enhancement of the distal internal auditory canals. Pet. Ex. 17 at 70.

On July 26, 2007, petitioner had an evaluation with Dr. Robert B. Nadelman, an infectious disease specialist, regarding a possible Lyme disease diagnosis. Pet. Ex. 46 at 57. Dr. Nadelman noted that petitioner "has a complex illness. I am uncertain of the etiology. I am also uncertain whether if she has ever had Lyme disease . . . . It would be extremely unlikely for American Lyme disease not to have a positive IgG Western blot with all of her neurologic and rheumatologic findings . . . . Vasculitis of some sort seems to make the most sense as a unifying diagnosis." Pet. Ex. 46 at 57. Petitioner underwent a repeat lumbar puncture on August 9, 2007, which showed normal results. Pet. Ex. 3 at 5-6; Pet. Ex. 21 at 70-77.

Petitioner returned to Dr. Gruis on August 23, 2007, for a follow-up consultation. Pet. Ex. 17 at 114-17. Dr. Gruis noted that "an extensive work-up has been done excluding other associated neurological conditions (including multiple sclerosis and atypical Guillain-Barré syndrome) sometimes associated with seventh nerve palsies. This leaves viral neuritis as the likely cause for her seventh nerve palsies." Pet. Ex. 17 at 116.

Petitioner saw Dr. Rebecca M. Kuenzler, a neurologist, at the Cleveland Clinic on October 2, 2007, for an evaluation for facial weakness. Pet. Ex. 12 at 7. Dr. Kuenzler noted that petitioner received an HPV vaccination in February 2007, and that petitioner was concerned that her symptoms were a reaction to the vaccine. Id. at 6. Dr. Kuenzler stated that petitioner's "history is most compatible with a Miller-Fisher variant of Guillain-Barré syndrome. The CSF did not show the typical albuminocytologic dissociation, but overall this is the best fit. I do not see a GQ 1 b antibody being sent.[9] This condition could have been related to her Gardasil vaccination, though this cannot be proven exclusively." Pet. Ex. 12 at 8.

Petitioner also saw Dr. Steven K. Schmitt, an infectious disease specialist at the Cleveland Clinic, on the same day, October 2, 2007. Pet Ex. 12 at 2-5. Dr. Schmitt noted that petitioner received the HPV vaccine in February 2007, and that she had facial nerve palsy with an acute onset afterwards. Id. He stated that the "[h]istory and testing data support a diagnosis of Guillain-Barré syndrome, Miller-Fis[]her variant. ... Clinically, [I] wonder about sarcoidosis (joints, facial palsy) as alternative diagnosis - though we have little supporting lab and radiographic data for this either." Id. at 4. Dr. Schmitt further stated that he "reviewed publically availably FDA data. There are around 15-20 reports of neurologic complications of HPV vaccine in adults so far, so such a reaction is possible in this patient with a temporally-related vaccination." Id.

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[9] Although Dr. Kuenzler stated she did not see that the GQ 1 b antibodies lab test was ordered, this test was ordered and the results were negative. See Pet. Ex. 11 at 20-21.

On October 15, 2007, Dr. Schechter noted that the physicians at the Cleveland Clinic felt that petitioner had "possible Guillain-Barré [Miller-Fisher] variant." Pet. Ex. 3 at 19. A neuro-ophthalmic exam by Edward M. Cohn, MD, on October 22, 2007, noted a resolving seventh nerve palsy and paresthesia in the region of her left cheek. Dr. Cohn stated that petitioner had associated her symptoms to her receipt of the HPV vaccine. Pet. Ex. 38 at 14-15. Another MRI of the brain was performed on petitioner on December 7, 2007, showing stable enhancement along the seventh nerve since the June 19, 2007 study, but the enhancement also appeared decreased when compared to a study performed on May 30, 2007. Pet. Ex. 2 at 7-8; Pet. Ex. 3 at 3-4. Petitioner underwent a SPECT brain scan on December 28, 2007, that did not show significant interval changes when compared to a study performed on December 15, 2004. Pet. Ex. 17 at 64.

In a report by Dr. Riutta, dated February 21, 2008, she noted that petitioner's condition was suggestive of an intermittent immune condition most consistent with chronic inflammatory demyelinating polyneuropathy. Pet. Ex. 2 at 13-15. During that same visit, petitioner inquired as to her initial diagnosis of Guillain-Barré syndrome. Dr. Riutta noted that the GBS diagnosis was made by Dr. Laban while petitioner was hospitalized, and that the only abnormalities identified by Dr. Riutta were consistent with bilateral cranial nerve VII palsy. Id. at 14. Petitioner also inquired whether her current condition could be related to her HPV vaccination that she received 42 days prior to her hospital admission in April 2007. Id. Dr. Riutta stated that petitioner's presentation met the criteria for an immune or vaccine-mediated process. Id.

On March 19, 2008, petitioner saw Dr. Schechter in a follow-up visit. Pet. Ex. 3 at 15. Dr. Schechter noted that petitioner "wonders if Gardasil has induced some of her symptoms," but stated that her symptoms "could be related to her prior history of Lyme disease. She is on IVIG, which is helping, Mobic and Lyrica." Id.

C. Petitioner's Ongoing Symptoms and Diagnosis

On April 20, 2011, petitioner presented to Robert C. Erickson, M.D., an ophthalmologist, who noted that petitioner continued to have facial paralysis since having GBS. Pet. Ex. 38 at 3. He also noted left facial weakness, double vision, vertical diplopia, and ocular hypertension. Id.

Petitioner was examined by Steven A. Telian, M.D., a neurologist, on May 24, 2011. Pet. Ex. 38 at 1. Dr. Telian noted that petitioner had a normal ear exam and that her facial nerve function was normal on the right and Grade 3/6 on the left, with residual spasm and synkinesis. Id. It was noted that petitioner received the HPV vaccine in 2007, and that IVIg therapy had been stopped in 2010. Id. Dr. Telian noted that he expected no progression of hearing loss. Id.

On May 8, 2013, petitioner underwent an EMG/NCV study because of complaints of progressive weakness on the left side of her face with numbness and tingling. Pet. Ex. 55 at 3-4. It was noted that petitioner had marked asymmetry between the two facial sides. Id. at 4. The nerve conduction study and EMG results were noted as being abnormal and consistent with an axonal lesion of the left facial nerve with chronic denervation. Id. at 4.

On July 3, 2013, Dr. Schechter ordered a second EMG/NCV of petitioner's bilateral facial motor nerves which showed prolonged distal latencies and low amplitudes bilaterally, worse on the left, consistent with bilateral facial neuropathy. Pet. Ex. 55 at 5-6.

At the hearing, petitioner testified that she still struggles to speak. Tr. at 6. She speaks with one side of her mouth because of bilateral facial nerve damage. Id. Petitioner also stated that she has permanent nerve damage of her face and eyes. Id. She explained that her eyes "do not track in tandem anymore." Id. at 7. Petitioner stated that she wears hearing aids because her auditory nerves were damaged. Id. at 8. In a note from her ophthalmologist, Dr. Erickson, dated April 29, 2013, he states that petitioner has had an incomplete recovery from her Miller-Fisher variant of GBS and that her residual injuries include facial paralysis (bilaterally, worse on the left side), delayed eyelid closure on the left, chronic eye irritation (worse on the left), delayed eye movement, intermittent double vision, and reduced reading stamina. Pet. Ex. 53 at 1. Although impaired by her injuries, petitioner testified that she graduated from law school in May 2013, and took the bar exam in July 2013. Id. at 22.

V. Expert Testimony and Analysis

A. Standards of Adjudication for a Causation Claim

To receive compensation under the Vaccine Act, petitioner must prove either (1) that she suffered a "Table Injury" – i.e., an injury falling within the Vaccine Injury Table – corresponding to one of the vaccinations in question, or (2) that her injury was actually caused by a vaccine (a "non-Table injury"). See §§ 300aa-13(a)(1)(A), 11(c)(1); § 300aa-14(a) as amended by 42 C.F.R. § 100.3; 300aa-11(c)(1)(C)(ii)(I); see also Moberly v. Sec'y of Health & Human Servs., 592 F.3d 1315, 1321 (Fed. Cir. 2010); Cappizzano v. Sec'y of Health & Human Servs., 440 F.3d 1317, 1320 (Fed. Cir. 2006). Since no table injury is alleged in this case, petitioner must prove causation in fact.

Petitioner bears the burden of demonstrating actual causation by a preponderance of the evidence. See Cedillo v. Sec'y of Health & Human Servs., 592 F.3d 1315, 1321 (Fed. Cir. 2010); § 300aa-13(a)(1). To do so, petitioner must provide: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between the vaccination and injury." Althen, 418 F.3d at 178. The preponderance of the evidence standard requires a petitioner to demonstrate that it is "more likely than not" that the vaccine caused her injury. Moberly, 592 F.3d at 1322 n.2. Proof of medical certainty is not required. Bunting v. Sec'y of Health & Human Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, petitioner must demonstrate that the vaccine was "not only [a] but for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Human Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); Pafford v. Sec'y of Health & Human Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The undersigned must consider the record "as a whole" and may not rule in petitioner's favor solely based on petitioner's own claims "unsubstantiated by medical records or medical opinion." § 13(a)(1).

Causation is determined on a case by case basis, with "no hard and fast per se scientific or medical rules." Knudsen v. Sec'y of Health & Human Servs., 35 F.3d 543, 548 (Fed. Cir. 1994). The Althen court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. Id. at 1279–80. The court also indicated that, in finding causation, the fact-finder may rely upon "circumstantial evidence," which the court found to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." Id. at 1280. In other words, any close calls regarding causation must be resolved in favor of the petitioner. Althen, 418 F.3d at 1280.

B. Expert Testimony

In Vaccine Act cases, expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 594-96 (1993); see also Cedillo, 617 F.3d at 1339 (citing Terran v. Sec'y of Health & Human Servs., 195 F.3d 1302, 1316 (Fed. Cir. 1999). "The Daubert factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." Terran, 195 F.3d at 1316 n.2 (citing Daubert, 509 U.S. at 592-95). In Vaccine Program cases, these factors are used in the weighing of the scientific evidence actually proffered and heard. Davis v. Sec'y of Health & Human Servs., 94 Fed. Cl. 53, 66–67 (Fed. Cl. 2010) ("uniquely in this Circuit, the Daubert factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"), aff'd, 420 F. App'x 923 (Fed. Cir. 2011). The flexible use of the Daubert factors to determine the persuasiveness and/or reliability of expert testimony in Vaccine Program cases has routinely been upheld. See, e.g., Snyder v. Sec'y of Health & Human Servs., 88 Fed. Cl. 706, 742–45 (2009).

Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." Broekelschen v. Sec'y of Health & Human Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing Lampe v. Sec'y of Health & Human Servs., 219 F.3d 1357, 1362 (Fed. Cir. 2000)). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the ipse dixit of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." Snyder, 88 Fed. Cl. at 743 (quoting Gen. Elec. Co. v. Joiner, 522 U.S. 146 (1997)). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. Moberly, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); see also Porter v. Sec'y of Health & Human Servs., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

Three experts testified at hearing: two for petitioner and one for respondent. The qualifications and testimony of each party's respective experts are summarized below.

1. Petitioner's Experts

a) Dr. Steven Schechter

(1) Medical background

Steven H. Schechter, M.D., is a neurologist and one of petitioner's treating physicians. Tr. at 31. He is board certified in neurology and is currently in private practice in West Bloomfield, Michigan. Tr. at 32. Dr. Schechter attended medical school at the Chicago Medical School in North Chicago, and graduated in 1987. Id. He then completed a one-year internship in internal medicine at Beaumont Hospital, and completed his residency in neurology at Henry Ford Hospital in Detroit. Id. at 33. Dr. Schechter then completed a fellowship in clinical neurophysiology at the University of Michigan. Id. After his fellowship, he went into private practice. Id. Dr. Schechter is currently on staff at Beaumont Hospital and an assistant professor of neurology at Oakland Beaumont Medical School. Id.

(2) History of petitioner's medical treatment

Dr. Schechter testified that he first saw petitioner on May 14, 2007, when she presented to him with complaints of facial weakness. Tr. at 32, 34. It was his understanding that petitioner had a complex of symptoms mainly consisting of a facial diplegia or bifacial weakness. Id. Over the years, Dr. Schechter stated that he continued to follow petitioner's medical history, including her claimed diagnosis of Lyme disease. He stated that he felt the diagnosis of Lyme disease was unlikely, but petitioner was left with a complex of systems that persisted. Tr. at 35.

(3) Diagnosis of petitioner's condition

Dr. Schechter offered an opinion regarding the nature of petitioner's injury based on his clinical experience with petitioner and a review of her treatment history. During his testimony, Dr. Schechter explained that the Miller-Fisher variant of GBS is "a clinical variance of Guillain-Barré syndrome which has distinct clinical features, ataxia,[10] areflexia,[11] and ophthalmoparesis."[12] Tr. at 65. He stated, however, that it is not always necessary to have the presence of all three features in a patient. Tr. at 39. Dr. Schechter explained that a patient may have "one or two of those features, but not necessarily have everything present." Id. In attempting to diagnosis petitioner's condition, Dr. Schechter testified that he originally considered a viral or post-viral etiology or a mechanism related to Lyme disease as a cause of petitioner's symptoms. Tr. at 36. However, after reviewing the records and speaking with an infectious disease specialist, Dr. Nadelman, who had also reviewed petitioner's medical history, Dr. Schechter felt that the diagnosis of Lyme disease could not be confirmed. Id. At that point, Dr. Schechter stated that he was left with a probable diagnosis of Guillain-Barré syndrome or a

[10] Ataxia is defined as "failure of muscular coordination; irregularity of muscular action." Dorland's, at 170.

[11] Ophthalmoparesis is defined as "paralysis of the eye muscles." Dorland's at 1329.

[12] Areflexia is defined as an "absence of reflexes." Dorland's at 130.

variant of Guillain-Barré (Miller-Fisher variant). Id.; Pet. Ex. 56 at 1-2. He stated that "given that [petitioner] had the cranial nerve involvement, she had vision complaints, I think that the Miller-Fisher variant of GBS is probably the most likely cause . . . ." Tr. at 39.

Dr. Schechter discussed the basis for his opinion that the Miller-Fisher variant of GBS was likely the proper diagnosis, as he explained that the cerebrospinal fluid can be normal with the Miller-Fisher variant of GBS during the first few days of the illness. Pet. Ex. 54 at 1. He further stated that the results of nerve conduction studies may also be normal, and F-wave responses (which are sometimes affected in GBS, Tr. at 70) may not be prolonged. Id.; Pet. Ex. 56 at 2. Dr. Schechter testified that petitioner had a symptom complex following her February 21, 2007 vaccination that was closest to a clinical definition for the Miller-Fisher variant of GBS. Tr. at 65-66. He also noted that he had ordered IVIg treatment for petitioner and that she had some improvement with the therapy. Dr. Schechter testified that "the improvement she had was consistent with the [Miller-Fisher] diagnosis; otherwise, why would she improve from it." Tr. at 71-72.

Dr. Schechter testified that he believed it was more likely than not that the Gardasil vaccine triggered petitioner's symptoms. In his report, Dr. Schechter stated that "it is more probable than not, that [petitioner's] facial diplegia and associated symptoms have resulted from GBS syndrome, or variant which presented in 2007, following Gardasil vaccine." Pet. Ex. 56 at 1-2. He further stated that "[g]iven the time course of vaccine followed by the onset of her clinical symptoms, with ongoing residual symptomatology, symptom complex may be consistent with a Miller-Fisher variant of GBS syndrome." Id. at 2. As he did during his testimony, Dr. Schechter described the symptom complex of the Miller-Fisher variant of GBS as including "opthalmoplegia, ataxia and areflexia." He explained that "[i]ncomplete forms of [Miller] Fisher Syndrome can be seen without ataxia or areflexia." Id. In describing petitioner's symptom complex, he stated that there was documentation of petitioner having right upper extremity clumsiness, left lower extremity clumsiness, bilateral Bell's phenomenon, asymmetry of the eyebrows bilaterally and slurred speech. Id. Dr. Schechter stated that the documentation "does support facial diplegia, skew deviation with residual, and other ongoing clinical symptoms. [Petitioner's] initial note did suggest there was some ataxia as well, based on extremity clumsiness. Reflexes may not always be lost in this syndrome." Id. at 3.

(4) Medical Theory

Dr. Schechter explained how Gardasil could have triggered petitioner's condition as his report states, "[i]t appears that the vaccine triggered an autoimmune type response resulting in a form of acute inflammatory demyelinating neuropathy, which has produced permanent symptoms which have persisted to this time. . . ." Pet. Ex. 54 at 2; Pet. Ex. 56 at 2. Dr. Schechter stated that "with a reasonable degree of medical certainty, [petitioner] appears to have sustained an immune mediated type of reaction which has left her with ongoing residual focal neurological deficits." Id. He stated that "based on the onset, the overall time course and the clinical symptoms that we have, which are not explained by any other mechanism," petitioner's condition was an immune-mediated reaction to the Gardasil vaccine. Tr. at 39.

(5) Logical Sequence of Cause and Effect

In his expert report, Dr. Schechter explained that the Gardasil vaccine "triggered an autoimmune type response resulting in a form of acute inflammatory demyelinating neuropathy, which has produced permanent symptoms which have persisted to this time . . . ." Pet. Ex. 54 at 2. Dr. Schechter noted that Dr. Laban had indicated that petitioner had a classic presentation of GBS with facial diplegia and bilateral cranial nerve conduction delays. Id. Dr. Schechter stated that the Beaumont emergency room records documented that petitioner had "right upper extremity clumsiness, left lower extremity clumsiness, bilateral Bell's phenomenon, asymmetry of the eyebrows bilaterally and slurred speech." Id. He also stated that the medical records noted that petitioner had "decreased sensation bilaterally of the facial area, sensory deficits, weakness, slurred speech, clumsiness of the left lower extremity, right upper extremity." Id. Based on these results, Dr. Schechter stated in his report that petitioner appeared to have sustained "an immune mediated type of reaction which has left her with ongoing residual focal neurological deficits." Pet. Ex. 54 at 3. He stated that these ongoing residual symptoms are "felt to have occurred following an immune mediated syndrome triggered by the Gardasil vaccine, for which she continues to suffer ongoing disability to this day, and felt to be permanent." Id. Regarding Lyme disease as being one of the alternate causes of petitioner's symptoms, Dr. Schechter stated that a number of petitioner's treating physicians have opined that petitioner's diagnosis of Lyme disease was "questionable at best." Id.

On cross examination, Dr. Schechter explained that petitioner had a complex medical history and clinical presentation, and that "there was a temporal time course where she had a clear onset of symptoms, a complex of symptoms following the vaccine, within that temporal time frame, it was helpful in terms of thinking about what the ultimate cause may be for her symptoms." Tr. at 59. Dr. Schechter also testified that there was no particular article or case study that led him to his opinion on causation in this case, but rather "there is evidence in the literature of... post-vaccine induced GBS, including Gardasil." Tr. at 60.

(6) Timing

Dr. Schechter stated that to a "reasonable degree of medical probability and/or certainty that the onset of petitioner's injury occurred during an appropriate temporal time period, that being between 1 week to 6 weeks." Pet. Ex. 57 at 1. He testified during the hearing that the timing can be variable, occurring "within a few days to several weeks." Tr. at 39.

(7) Review of the MRIs and EMGs

Petitioner underwent a number of MRIs prior to and soon after her Gardasil vaccination. The pre-vaccination MRIs include an MRI conducted on April 1, 2001 (not included in the record), a brain MRI conducted on October 28, 2002 (Pet. Ex. 17 at 97), and an MRA/MRI of petitioner's head conducted on October 15, 2003 (Pet. Ex. 17 at 96). The first MRI report that was filed in this case, an October 28, 2002 MRI, conducted on the brain, showed, as compared with a study performed on September 15, 2001 (not included in the record), "[t]he internal auditory canals and orbital contents are within normal limits . . . . There is no evidence for enhancing lesions within the brain, abnormal signal intensity within the brain parenchyma or orbits and the CSF spaces are unremarkable . . . . Impression: Unremarkable evaluation of the

brain and orbits without change since prior study." Pet. Ex. 17 at 97. Dr. Schechter testified that the results of this MRI appeared to be normal. Tr. at 86. The MRA conducted on October 15, 2003, also yielded normal results. Pet. Ex. 17 at 96; tr. at 86-87.

Post-vaccination MRIs/MRAs were conducted on April 5 and 14, 2007, May 30, 2007, June 19, 2007 and December 7, 2007. Tr. at 80. A brain CT scan was conducted on April 4, 2007, which showed normal results. Pet. Ex. 17 at 77. The April 14, 2007 MRI references an MRI/MRA that was conducted on April 5, 2007 (Pet. Ex. 10 at 155), for comparative purposes. Tr. at 90. The MRI report dated April 14, 2007, states that "there is mild, symmetric enhancement of the distal intracanalicular acoustic nerve complexes, the bilateral geniculate ganglia, and the descending portion of the facial nerves bilaterally. This is slightly more enhancement than would be expected for physiologic vascular enhancement." Tr. at 90; Pet. Ex. 10 at 155. Dr. Schechter testified that the description of enhancement on this MRI is consistent with his opinion that there was an acute inflammatory reaction occurring post-vaccination. Tr. at 92. According to Dr. Schechter, an enhancement on an MRI demonstrates a newer "active process" and "active inflammation" which means that there is a breakdown of the blood-brain barrier. Tr. at 81.

Dr. Schechter testified that the May 30, 2007 MRI conducted on petitioner's brain and internal auditory canal, as compared to the MRI performed on April 13, 2007, showed a stable appearance of the enhancement of the bilateral facial nerve, i.e., the seventh cranial nerve. Tr. at 80-81; Pet. Ex. 17 at 71. The December 7, 2007 MRI showed decreased enhancement which, according to Dr. Schechter, meant that the inflammatory process was "settling down." Tr. at 82; Pet. Ex. 17 at 66. In summary, Dr. Schechter testified that this enhancement process demonstrated a change in the MRIs before and after petitioner's Gardasil vaccination. Tr. at 87.

Regarding the facial nerve EMG study, Dr. Schechter testified that it is a test that is "done to check the integrity of the facial nerve." Tr. at 73. He explained that demyelination can affect the outer casing of a nerve, while at other times, "the actual axon or the wiring of the nerve is affected." Id. Dr. Schechter testified that in petitioner's case, "she had both demyelinating and axonal involvement, which suggests a more significant injury to the nerve with a poor prognosis in terms of recovery." Id. He explained that while the EMG study shows the damage to the facial nerve, it does not speak to the cause of the injury. Id.

b) Dr. Axelrod

(1) Medical Background

David Allen Axelrod, M.D., is a clinical immunologist trained at McGill University (Montreal) and the National Institutes of Health. Pet. Ex. 80 at 1. He obtained his medical degree at the University of Michigan Medical School. Tr. 96. Dr. Axelrod trained in internal medicine at the University of Toronto and then at William Beaumont Hospital. Tr. 96-97. He completed a fellowship in allergy, immunology and rheumatology at McGill University and another two years at the National Institutes of Health. Id. He was also a principal investigator at the Walter Reed Army Institute of Research (Bethesda) and his laboratory participated in vaccine development. Id. at 128. Dr. Axelrod currently holds a visiting faculty appointment at Penn State Hershey. Tr. 127-28.

(2) Diagnosis of Petitioner's Condition

Dr. Axelrod opinioned that petitioner suffered from either a de novo autoimmune demyelinating disorder or a recurrence/exacerbation of an underlying demyelinating disorder. Pet. Pre-hearing Memorandum at 1, 4; Pet. Ex. 23 at 3. During his testimony, he stated that he was not a neurologist and was relying on the medical records of petitioner's treating physicians regarding her diagnosis of the Miller-Fisher variant of GBS when reaching his opinions in this case. Tr. at 135-37.

(3) Medical Theory

Dr. Axelrod proposed the theory of molecular mimicry to explain how the HPV vaccine could cause GBS (including the Miller-Fisher variant of GBS). Tr. at 149. Molecular mimicry has been defined to be a "sequence and/or conformational homology between an exogenous agent (foreign antigen) and self-antigen leading to the development of tissue damage and clinical disease from antibodies and T cells directed initially against the exogenous agent that also react against self-antigen."[13] In his expert report, Dr. Axelrod explained that "[i]mmune reactions to vaccines depend upon the individual's immune system's ability to recognize the vaccine as a foreign substance." Pet. Ex. 23 at 3. The "human papillomavirus and the Gardasil vaccine contain structures, to which the human immune response reacts, to protect vaccinated individual[s], including the L1 and L2 proteins. Similar structures are present in the myelin of the nervous system. Damage to the myelin and astrocytes is a primary finding in autoimmune demyelinating disorders." Id. Dr. Axelrod explained that "the [HPV] virus contains structures that are similar to those in the human being and if you develop an immune response to those antigens which is, you know, how the vaccine works . . . . there's a chance that it can attack the normal tissue." Tr. at 106. As a result, the antibodies that were developed as an immune response to the vaccine begin to damage the normal structures in the human host, including possible damage to the nervous system. Id. at 118; Pet. Ex. 43.

To support his theory, Dr. Axelrod relies on an article by Wucherpfennig[14] which demonstrates that there is "some structural homology between the vaccine peptides and a portion of the myelin basic protein. And that portion of the myelin basic protein could cause cells from human beings to respond when they were stimulated with that segment . . . . in other words, the cells – cell receptors and antibody receptors recognize the structures on myelin as they recognize the structures on the Gardasil vaccine" and cause damage which may lead to various injuries and diseases. Tr. at 107.

On cross-examination, Dr. Axelrod was asked whether there were any animal models to support his theory that the HPV vaccine or any component of the HPV vaccine could cause either GBS or the Miller-Fisher variant of GBS. Tr. at 143. Dr. Axelrod explained that he would not expect to see any animal studies on those specific issues because the increase in risk would

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[13] Institute of Medicine, Adverse Effects of Vaccines: Evidence and Causality at 70 (Stratton K. et al., eds. 2011) [hereinafter "Adverse Effects of Vaccines"].

[14] Pet. Ex. 37 (Kal W. Wucherpfenning et al., Molecular Mimicry in T Cell-Mediated Autoimmunity: Viral Peptides Activate Human T Cell Clones Specific for Myelin Basic Protein, 80 Cell 695-705 (1995).

be "incredibly small" and it would "be very hard to have enough sample sizes of anything to sort that out, which is . . . . why the epidemiologic studies don't help us." Id.

(4) Logical Sequence of Cause and Effect

Dr. Axelrod testified that based on the timing of petitioner's onset of symptoms and her receipt of the Gardasil vaccination, "it made sense" from an immunologic standpoint, that the vaccine contributed to petitioner's injuries as has been reported in the medical literature. Tr. at 108-09. And because "there were antigens – structures on the vaccine that were similar to structures on the human body. And if the – you make an immune response to those same structures that are common to both, you may end up with damage to the normal tissues in the human being." Id. at 109. Dr. Axelrod testified that he also reached his opinion because other causes of petitioner's condition, such as Lyme disease and the negative results from laboratory tests for viruses and bacteria, had been ruled out. Tr. at 150.

When asked about what type of reaction he would expect to see, as an immunologist, when a reaction occurs in response to a vaccine, Dr. Axelrod testified that he would expect to see the development of a problem, such as an inflammatory response, and then he would expect that problem to "taper off" unless the problem caused permanent damage. Tr. at 150-51. He stated, however, that there was no way to measure whether petitioner had an inflammatory reaction to the Gardasil vaccine in this case. Id. Dr. Axelrod testified that at one point in time, there was an improvement to petitioner's clinical course that was documented in the records, and this provided evidence of petitioner's clinical course that was consistent with his theory. Id. at 152.

When questioned about petitioner's prior symptoms, which have indicated that there was possibly a prior immune process occurring, Dr. Axelrod testified that the presence of that process would not change his opinion. Tr. at 156. His opinion would be that the vaccine exacerbated any preexisting condition. Id.

(5) Timing

In his expert report, Dr. Axelrod stated that "[a]t least 14 days may be required for a vaccine to produce a measurable primary or secondary immune response, which may be followed by the development of disease. In fact, Guillain-Barré Syndrome may occur up to at least 6 weeks, following vaccination with Gardasil." Pet. Ex. 80 at 2-3. Dr. Axelrod similarly testified that it would take at least two to three weeks to look at an immune response to a vaccination, such as Gardasil. Tr. at 105-06. He testified that at least one of the articles he cited in his report that discussed vaccine reaction stated that the reaction occurred six weeks later. Tr. at 146. And because this was petitioner's first and only Gardasil vaccine (she did not receive the boosters), he testified that such a reaction "will take longer." Tr. at 147. Dr. Axelrod stated that the expected response for a secondary reaction is much sooner than for a primary reaction. Tr. at 147-48.

(6) Review of the MRIs and EMGs

Dr. Axelrod testified that he does not interpret MRI films. Tr. at 153. He stated that if the EMG studies and MRIs/MRAs showed demyelination, it would be consistent with his theory that an immune response had occurred. Id. at 154.

2. Respondent's Expert – Dr. Thomas Leist

(1) Medical Background

Dr. Thomas Leist is a neuroimmunologist and is currently employed at the Thomas Jefferson University in Philadelphia, Pennsylvania. Tr. 160; Resp't Ex. O. He holds a doctoral degree in biochemistry and immunology from the University of Zurich. Id. Dr. Leist completed fellowships in immunology and virology, both at the University of Zurich and at UCLA. Tr. at 160. He received his medical degree from the University of Miami. Id. Dr. Leist completed his residency in neurology at Cornell Medical Center, Sloan Kettering Memorial Cancer Center. He held a position as Senior Clinical Staff Associate with the National Institute of Neurological Disorders and Stroke at the National Institutes of Health. Dr. Leist is an attending physician and works with fellows and residents. Tr. 115. He is board-certified in psychiatry and adult neurology and serves as an editor and peer-reviewer for medical journals. Tr. 161; Resp't Ex. O at 1. Currently at Thomas Jefferson University, Dr. Leist is a professor of neurology and directs the clinical and clinically-based research efforts in multiple sclerosis. Tr. 160-61. Dr. Leist states that he has treated patients with GBS and patients with the Miller-Fisher variant of GBS. Tr. at 161-62.

(2) Diagnosis of Petitioner's Condition

Dr. Leist opined that petitioner did not suffer from GBS, a variant of GBS, or a demyelinating injury, and that an appropriate diagnosis was never confirmed in her case. Tr. at 163-65. Dr. Leist stated that he relied on the medical records to reach his opinion, including the contemporary medical records from Dr. Schechter. Tr. at 164. Dr. Leist noted that petitioner had evidence of normal deep tendon reflexes and an absence of ataxia, ophthalmoplegia, and areflexia. Id. Dr. Leist stated in his report that Dr. Schechter first saw petitioner on May 14, 2007, or one month following her April 2007 hospitalization. During that exam with Dr. Schechter, petitioner had normal extremity strength, sensation, and deep tendon reflexes. Dr. Leist felt that petitioner's facial weakness was possibly post-viral or related to Lyme disease. Resp't Ex. A at 15-16.

(3) Response to Petitioner's Proposed Medical Theory

Regarding petitioner's theory of molecular mimicry, Dr. Leist states that "it is always possible to find sequence homologies, short sequence homologies between peptides. . . . the mere occurrence of these homologies doesn't, in itself, indicate that such a homology will give rise to a cross-reactive immune response." Id. Dr. Leist explained that the human papillomavirus is not recognized as a source of demyelinating illness or as a cause of demyelinating illness. Tr. at 181. And, "there is no evidence that human papillomavirus, as an intact infectious virus, causes demyelinating disease" or "is associated with demyelinating disease." Id. In reviewing the

Kanduc[15] article, Dr. Leist stated that the authors of the article do not "go beyond describing sequence homologies. So, this article doesn't, in itself, step beyond describing the fact that, as we already mentioned. . . . that obviously if you look through the total of genomes of all the organisms on this globe, you will find homologies." Tr. at 183. Regarding the human papillomavirus specifically, Dr. Leist stated that the Kanduc paper "falls under the premise as outlined in the IOM report that a sequence homology, in itself, does not provide proof of a biologically important reaction as a matter of this sequence homology." Id.

Dr. Leist conducted an extensive critique of the other medical literature on which petitioner's experts relied. In his view, none of the medical literature supports petitioner's theory that the HPV vaccine can cause GBS or the Miller-Fisher variant of GBS via molecular mimicry. See Resp't Ex. A at 11-12.

(4) Response to Petitioner's Logical Sequence of Cause and Effect Argument

Dr. Leist also testified that he believed that the injuries that petitioner alleges were caused by her February 21, 2007 Gardasil vaccine all pre-date her vaccination. Tr. at 168. He stated that the medical records document petitioner's hearing loss/impairment as early as 2001. Tr. at 168-69. Dr. Leist also testified that the medical records document petitioner's facial weakness as early as 2001 with worsening in 2003 and 2004. Id. And he also noted that petitioner's complaints of visual disturbances all preceded her Gardasil vaccination. Id. It is Dr. Leist's opinion that the symptoms and the process that led to petitioner's facial weakness after her February 2007 Gardasil vaccine, were the same that led to her facial weakness prior to the vaccination. Tr. at 170. He also hypothesizes that petitioner may have had a foodborne illness shortly after the February 2007 vaccination, which may have contributed to the increase of symptoms shortly after vaccination. Id.

(5) Response to Petitioner's Timing Argument

Regarding the timing issue, Dr. Leist opined that a time interval of over 40 days between vaccine administration and the occurrence of symptoms further weighs against the vaccine as a cause of petitioner's clinical presentation. Resp't Ex. A at 13. However, he admitted during his testimony that if the proof of an appropriate temporal association is extrapolated from studies involving the swine flu vaccine, although on the outer limits, the 41 day time period between Gardasil vaccination and onset of symptoms in petitioner's case would fall within the appropriate time frame. Tr. at 189. Dr. Leist testified, however, that the likelihood of vaccine causation goes down from the fourth to sixth week after vaccine administration. Id.; tr. at 213-14.

In summary, Dr. Leist stated that the bases for his opinion are: (1) that petitioner had a history of similar medical events occur prior to her Gardasil vaccination, (2) that forms of the human papillomavirus are not recognized as commonly associated with Guillain-Barré syndrome, (3) that petitioner's negative anti-ganglioside antibodies and her cerebrospinal fluid findings were not supportive of Guillain-Barré syndrome or the Miller-Fisher variant, and (4)

[15] Pet. Ex. 44 (Darja Kanduc, Quantifying the Possible Cross-Reactivity Risk of an HPV16 Vaccine 8 J Experimental Therapeutics and Oncology 65-76 (2009))

that the time interval between petitioner's vaccination and the onset of petitioner's symptoms was too long. See Resp't Ex. A at 16.

(6) Review of the MRI/MRA and EMG Findings

In his written reports, Dr. Leist stated that petitioner's MRI films did not show evidence of a demyelinating injury post-vaccination, and that the pre- and post-vaccination MRI films he reviewed showed comparable, non-specific findings. Resp't Ex. G at 2. Dr. Leist stated that in reviewing petitioner's MRIs, the "absence of new lesions in the brain parenchyma and the lack of motor and sensory findings below the neck essentially rule out diagnoses of a demyelinating central nervous system disorder including ADEM, transverse myelitis, and MS." Resp't Ex. A at 15.

During his testimony, however, Dr. Leist offered a slightly different opinion. Dr. Leist confirmed that he also reviewed and interpreted the MRI films in addition to the MRI reports. Specifically, in reviewing each MRI, Dr. Leist testified that in petitioner's April 1, 2001 brain MRI, he found some "nonspecific white matter changes" which are indicative of "small vascular injury" but are not "emphatically indicative or supportive of [petitioner's] disease process." Tr. at 172. With the October 28, 2002 MRI study, Dr. Leist testified that he found "comparable abnormalities or signals." Tr. at 173. The October 15, 2003 MR angiogram was a "normal MR angiogram." Id. For the post-vaccination MRIs, Dr. Leist testified that the April 5, 2007 MRI, which was compared to two older studies, showed the same "signal abnormalities." Tr. at 174. He stated that he did not see any significant change to the prior studies. Id. However, Dr. Leist clarified that this study "was not a study done properly for cranial nerves." He agreed that there was "the presence of some vasculature . . . . or has some enhancement because blood vessels are there . . . . I didn't see – or, to me, to my eye, this didn't look like significant enhancement." Tr. at 175. Dr. Leist testified regarding the April 14, 2007 MRI, that if the "verdict to be true that there is enhancement, that would be obviously an indication that there is a very longitudinal inflammatory process ongoing in these nerves." Tr. at 178. In his report, Dr. Leist referred to the June 19, 2007 MRI report which interpreted the MRI "as showing enhancement of the distal internal auditory canals extending to the geniculate ganglion and the descending portions of the facial nerve." Resp't Ex. A at 8; Pet. Ex. 17 at 70.

C. Analysis of the Causation Claim

A threshold issue in this case is whether petitioner had GBS. A determination of what afflicted petitioner "is a prerequisite to ... [a causation] analysis." Broekelschen, 618 F.3d at 1346. For the reasons discussed below, the undersigned finds by a preponderance of the evidence that petitioner did have the Miller-Fisher variant of GBS.

a) Dispute over Petitioner's Diagnosis

The preliminary questions to be resolved are: (1) whether petitioner suffered from the Miller-Fisher variant of GBS or an autoimmune demyelinating disorder after her receipt of the Gardasil vaccine on February 21, 2007, and (2) whether petitioner suffered from an identifiable, underlying medical condition before February 21, 2007. The undersigned finds that petitioner suffered from the Miller-Fisher variant of GBS after her receipt of the Gardasil vaccine on

February 21, 2007. The undersigned further finds that petitioner suffered from an illness or injury prior to February 21, 2007, although that illness or injury was not clearly identified and appears to be separate and distinct from her development of Miller-Fisher GBS. Further, the undersigned finds that an exact diagnosis or identification of petitioner's prior illness is not necessary for the purpose of finding causation in this entitlement decision.

In determining petitioner's diagnosis, the undersigned reviewed and relied on statements in the medical records, as medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the patient. Cucuras v. Sec'y of Health & Human Servs., 993 F.2d 1525, 1528 (Fed. Cir.1993). In addition, the opinions of petitioner's treating physicians are "quite probative" as treating physicians are in the "best position" to diagnose and determine the cause of petitioner's condition. Capizzano, 440 F.3d at 1326. However, medical records setting forth a treating physician's views do not per se bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. § 300aa–13(b)(1); Snyder, 88 Fed. Cl. at 745 n.67. The views of treating physicians should also be weighed against other, contrary evidence present in the record—including conflicting opinions among the treating physicians themselves. Hibbard v. Sec'y of Health & Human Servs., 100 Fed. Cl. 742, 749 (Fed. Cl. 2011), aff'd, 698 F.3d 1355 (Fed. Cir. 2012); Caves v. Sec'y of Health & Human Servs., 100 Fed. Cl. 119, 136 (Fed. Cl. 2011), aff'd, 463 F. App'x 932 (Fed. Cir. 2012); Veryzer v. Sec'y of Health & Human Servs., No. 06–522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), aff'd, 100 Fed. Cl. 344 (2011).

b) Did petitioner suffer from the Miller-Fisher variant of Guillain-Barré syndrome or an auto-immune demyelinating disorder after February 21, 2007?

Upon careful examination of petitioner's medical records, the undersigned notes that petitioner's treating physicians did not reach a consensus in reaching a diagnosis for petitioner. However, the undersigned finds that the medical records and the testimony of the respective experts suggest that petitioner more likely than not suffered from the Miller-Fisher variant of GBS.

(1) Guillain-Barré Syndrome and the Miller-Fisher Variant

A brief description of Guillain-Barré Syndrome (GBS) is helpful to understand the Miller-Fisher variant of the condition. Clinically, GBS is characterized by the acute or subacute onset of varying degrees of weakness in limbs associated with hypo- or areflexia, and a characteristic profile in the cerebrospinal fluid. See Resp't Ex. N at 4. Patients typically experience progressive limb weakness, most often beginning in the legs and ascending to the arms and bulbar muscles. Id. The weakness is associated with decreased or absent deep tendon reflexes, and tends to be relatively symmetric. Id. The weakness progresses in an acute to subacute fashion, reaching its clinical nadir of weakness within two to four weeks, although in some cases rapidly progressive weakness reaching nadir within several hours may be seen. Id. Cranial nerve palsies, including involvement of the facial nerve resulting in facial weakness or extraocular motor nerve involvement or bulbar palsy may be seen. In a small percentage of cases, particularly if CSF is obtained early in the course of illness, CSF protein may be normal. Id.

The Miller-Fisher variant of GBS is a "clinical syndrome characterized by a triad of ataxia, ophthalmoplegia, and areflexia . . . ." Resp't Ex. N at 7. While the "classic triad is often clinically recognized and occurs in the absence of limb weakness, in some cases there is clinical overlap with GBS, with limb weakness present; such cases are considered to be GBS-[Miller-Fisher syndrome] overlap syndromes. Certain features of Miller-Fisher syndrome, including the general interval between onset and clinical nadir and presence of cytoalbuminologic dissociation,[16] are similar to that for GBS. In general, electrodiagnostic findings are normal, or abnormalities are limited to sensory nerves.

(2) Petitioner's Evidence

A review of petitioner's records demonstrates that the range of possible diagnoses considered by her treating physicians included the Miller-Fisher variant of GBS, Lyme disease, chronic inflammatory demyelinating polyneuropathy, and a viral neuritis. While petitioner did not present with the classic triad of symptoms for the Miller-Fisher variant, at least five of petitioner's treating physicians considered her history and clinical presentation of symptoms, and either considered the Miller-Fisher variant of GBS as a diagnosis or actually diagnosed petitioner with that condition. In a consultation on April 11, 2007, Dr. Ernstoff, a neurologist, considered a possible diagnosis of Miller-Fisher syndrome as a diagnosis. Pet. Ex. 10 at 155-56. On April 12, 2007, Dr. Laban stated that petitioner had a "classic presentation of Guillain-Barré [] syndrome of acute bilateral facial paresis." Pet. Ex. 4 at 340. In October 2007, after reviewing petitioner's medical records and history, Dr. Kuenzler at the Cleveland Clinic assessed that petitioner's "history is most compatible with a Miller-Fisher variant of Guillain-Barré syndrome." Pet. Ex. 12 at 8. Also in October 2007, Dr. Schmitt noted that petitioner's "[h]istory and testing data support a diagnosis of Guillain-Barré syndrome, Miller-Fis[]her variant." Pet Ex. 12 at 2-5. On October 15, 2007, Dr. Schechter noted that the physicians at the Cleveland Clinic felt that petitioner had "possible Guillain-Barré [Miller-Fisher] variant." Pet. Ex. 3 at 19.

In addition to the medical records, petitioner's expert and treating neurologist, Dr. Schechter, testified at the hearing and stated that to "a reasonable degree of certainty" the most compatible diagnosis with petitioner's symptoms was the Miller-Fisher variant of GBS. Tr. at 60. In supporting his diagnosis, Dr. Schechter stated that petitioner had coordination difficulties, vision complaints and double vision, which could be related to the ophthalmoparesis aspect, but he agreed that petitioner did not have ataxia. Tr. at 77-78. Dr. Schechter also ordered IVIg treatment for petitioner and noted that she had some improvement with the therapy. Dr. Schechter testified that "the improvement she had was consistent with the [Miller-Fisher] diagnosis; otherwise, why would she improve from it." Tr. at 71-72.

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[16] Cytoalbuminologic dissociation is defined as "an elevation of CSF protein levels (above normal reference values for the laboratory doing the testing) in the relative absence of pleocytosis (elevation of CSF WBC [white blood cell count]). Based upon the best available evidence, the Working Group has used a CSF WBC cutoff value of <50 WBC / μl for what would be consistent with GBS. It is recognized that in some cases of otherwise clinically typical GBS, CSF may be 'normal', particularly if obtained within the first week of illness." Resp't Ex. N at 9.

On cross examination, Dr. Schechter agreed that petitioner did not present with all of the classic features of the Miller-Fisher variant. Tr. at 66. However, he still concluded that petitioner sustained a new insult after her Gardasil vaccination and felt that the best diagnosis for petitioner's condition was Miller-Fisher syndrome based on his knowledge of petitioner and her case. Id. at 60, 66.

As stated above, Dr. Axelrod testified that he was not a neurologist and was relying on the medical records of petitioner's treating physicians regarding the diagnosis of the Miller-Fisher variant of GBS when reaching his opinions in this case. Tr. at 135-37.

(3) Respondent's Evidence

Dr. Leist opined that petitioner did not suffer from GBS, a variant of GBS, or a demyelinating injury, and that an appropriate diagnosis was never confirmed in her case. Tr. at 163-65. Dr. Leist stated that he relied on the medical records to reach this opinion, including the contemporary records from Dr. Schechter that document findings undermining a diagnosis of the Miller-Fisher variant of GBS. Tr. at 164. Dr. Leist noted that petitioner had negative anti-ganglioside antibodies and unsupportive CSF findings. She also had evidence of normal deep tendon reflexes and an absence of ataxia, ophthalmoplegia, and areflexia. Dr. Leist notes in his report that Dr. Schechter first saw petitioner on May 14, 2007, or one month following her WBH discharge. At that visit, Dr. Schechter made no mention of symptoms of extremity weakness. He noted that petitioner had normal extremity strength, sensation, and deep tendon reflexes on exam. He felt that her facial weakness was possibly post-viral or related to Lyme disease.

Regarding the lack of evidence to support a demyelinating injury, Dr. Leist noted that petitioner's MRI films did not show evidence of a demyelinating injury post-vaccination, and that the pre- and post-vaccination films he reviewed showed comparable, non-specific findings. During the hearing, however, Dr. Leist agreed that petitioner did show evidence of bilateral axonal injury to her facial nerve (a loss of nerve cells) and demyelination after the February 21, 2007 Gardasil vaccine as shown by Dr. Schechter's EMG. Tr. at 191-93. Dr. Leist goes on to state that "obviously there was facial weakness and that there was an exacerbation or there was a worsening of the facial weakness," but he states that there is no way to date when that injury occurred, although he does agree that a worsening of the facial nerve injury occurred in April 2007. Id. at 193-94.

(4) Evaluation of the Evidence

The undersigned must consider the record as a whole in evaluating petitioner's injury. § 13(a)(1). Here, the record and testimony supports the conclusion that petitioner more likely than not suffered from Miller-Fisher syndrome after February 21, 2007. This finding is informed by the medical records and the opinions of petitioner's treating physicians, a number of whom diagnosed petitioner with the Miller-Fisher variant of GBS. See Cappizzano, 440 F.3d at 1326. It is also further informed by the medical literature submitted by the parties, which supports petitioner's diagnosis.

In reviewing the medical records, the undersigned notes that although no diagnosis was agreed upon at the time, there is no question that petitioner suffered a significant injury after her

Gardasil vaccination which led to a 12-day hospitalization. The record contains documented complaints of petitioner's vision difficulties, coordination difficulties, and bilateral facial weakness after her Gardasil vaccination. See Pet. Ex. 3 at 25; Pet. Ex. 10 at 58; Pet. Ex. 14 at 6; Pet. Ex. 17 at 156-57; Pet. Ex. 19 at 6-7, 17-18. Petitioner was diagnosed with facial nerve palsy of the seventh cranial nerve, which resulted in facial weakness. Pet. Ex. 10 at 109. At least five of petitioner's treating physicians considered her history and clinical presentation of symptoms, and either considered the Miller-Fisher variant of GBS as a diagnosis or actually diagnosed petitioner with that condition. All of these facts, in addition to the testimony and reports of the parties' experts support a finding that petitioner suffered from the Miller-Fisher variant of GBS. Pet. Ex. 17 at 75-76.

Because the undersigned has found that the evidence supports the conclusion that petitioner suffered from the Miller-Fisher variant of GBS after February 21, 2007, the second issue in dispute, whether petitioner suffered an auto-immune demyelinating disorder after her Gardasil vaccination is answered in the affirmative.

c) Did petitioner suffer from an identifiable, underlying medical condition before February 21, 2007?

The parties ask the special master to determine whether petitioner suffered from an "identifiable, underlying medical condition before February 21, 2007." Based on the a review of the records filed in this case, the undersigned finds that petitioner did suffer from some symptoms and injuries prior to February 21, 2007, although the exact diagnosis of her condition was never agreed upon by all of petitioner's treating physicians and remains unclear. However, the undersigned is not required to diagnose petitioner's condition. In Lombardi, the Federal Circuit stated "[T]he function of a special master is not to 'diagnose' vaccine-related injuries, but instead to determine 'based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [petitioner's] injury.' " Lombardi v. Sec'y of Health & Human Servs., 656 F.3d 1343, 1351 (Fed. Cir. 2011) (citing Andreu v. Sec'y of Health & Human Servs., 569 F.3d 1367, 1382 (Fed. Cir. 2009). Furthermore, the Althen analysis could be applied even if petitioner did not have a specific diagnosis because there is no affirmative burden on petitioner to establish a specific diagnosis. See Kelley v. Sec'y of Health & Human Servs., 68 Fed. Cl. 84, 100 (2005) ("The Vaccine Act does not require petitioners coming under the non-Table injury provision to categorize their injury; they are merely required to show that the vaccine in question caused them injury – regardless of the ultimate diagnosis.")

In reviewing petitioner's medical records just prior to her vaccination of February 21, 2007, the undersigned notes that petitioner appeared to be doing well with only minor complaints noted. During an exam conducted at the Michigan Ear Institute on October 9, 2006, it was noted that petitioner did have some slight facial weakness on her right side. Pet. Ex. 19 at 16. Petitioner's physical exam was otherwise normal with the exception of some slight instability noted on her balance tests. Id. An examination of petitioner's eyes revealed "globes normal; extraocular muscles intact." Id. An exam on October 30, 2006, at the Michigan Ear Institute noted that petitioner had a "healthy appearance," that she was "alert and oriented," and that her facial function was normal. Id. at 16. Some degree of hearing loss in both ears was also noted, and petitioner was wearing a hearing aid on both sides. Pet. Ex. 19 at 16; Pet. Ex. 10 at 223-24.

The last medical record filed prior to petitioner's February 21, 2007 vaccination, is a record from her gynecologist dated November 20, 2006. Pet. Ex. 11 at 3. It is noted that petitioner is "doing well" and that she had "no new medical problems." Id. [redacted]

Although the undersigned finds that petitioner did suffer from some symptoms and injuries prior to February 21, 2007, the undersigned is not assigning a specific diagnosis to that condition. Furthermore, because the undersigned finds that petitioner suffered from a de novo onset of the Miller-Fisher variant of GBS after her February 21, 2007 Gardasil vaccination, petitioner has sufficiently proven a causation-in-fact claim, and the analysis for the alternative significant aggravation theory pled by petitioner in her post-hearing brief is unnecessary.

d) Application of Althen Prongs

(1) Prong One: Can the HPV Vaccine Cause the Miller-Fisher Variant of GBS and/or an Autoimmune Demyelinating Disorder?

Under Althen prong one, petitioner must provide a "reputable medical theory" demonstrating that the vaccine can cause the type of injury alleged. Pafford, 451 F.3d at 1355-56 (citations omitted). To satisfy this prong, petitioner's theory must be based on a "sound and reliable medical or scientific explanation." Knudsen, 35 F.3d at 548. The medical theory only need be "legally probably, not medically or scientifically certain." Id. at 549. A petitioner may satisfy Althen prong one without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a theory that has general acceptance in the medical or scientific community. Andreu, 569 F.3d at 1378-79 (citing Cappizzano, 440 F.3d at 1325-26).

As described above, petitioner presented a theory of molecular mimicry to explain how the Gardasil vaccine could cause GBS (including the Miller-Fisher variant of GBS). Petitioner's expert, Dr. Axelrod, explained that the human papillomavirus and the Gardasil vaccine contain structures, to which the human immune response reacts, to protect vaccinated individuals. As Dr. Axelrod explained, the human papillomavirus and the Gardasil vaccine contains structures, to which the human immune response system reacts, to protect vaccinated individuals, including L1 capsid protein. If an individual develops antibodies from the vaccine to these same structures, there may be damage to the normal structures within the nervous system. Pet. Ex. 23 at 3. Dr. Schechter presented a more generalized theory stating that "with a reasonable degree of medical certainty, [petitioner] appears to have sustained an immune mediated type of reaction which has left her with ongoing residual focal neurological deficits." Pet. Ex. 54 at 2; Pet. Ex. 56 at 2.

Dr. Leist did not specifically oppose the molecular mimicry theory as a plausible theory other than to state that there is no evidence to demonstrate that molecular mimicry plays a role in explaining how the Gardasil vaccine specifically can cause GBS or Miller-Fisher syndrome. Dr. Leist explained that although there may be homology between the components of the vaccine and part of the human body, there is no evidence to demonstrate that an autoimmune condition is likely to occur.

The undersigned finds that petitioner has provided preponderant evidence that the Gardasil vaccine can cause GBS (or the Miller-Fisher variant) via molecular mimicry. Accordingly, petitioner has satisfied Althen Prong One.

(2) Prong Two: Did the HPV Vaccine Cause Petitioner's GBS and/or Demyelinating Disorder?

The second Althen prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. Althen, 418 F.3d at 1278; Andreu, 569 F.3d at 1375–77; Capizzano, 440 F.3d at 1326; Grant, 956 F.2d at 1148. In evaluating whether this prong is satisfied, the opinions and views of the injured party's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury' ") (quoting Althen, 418 F.3d at 1280). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the patient. Cucuras, 993 F.2d at 1528. Petitioner need not make a specific type of evidentiary showing, i.e., petitioner is not required to offer "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. See id. at 1325-26.

In his expert report, Dr. Schechter stated that "[g]iven the time course of the vaccine followed by the onset of her clinical symptoms, with ongoing residual symptomatology, [petitioner's] symptom complex may be consistent with a Miller-Fisher variant GBS syndrome." Pet. Ex. 54 at 2. He explained that petitioner had a complex medical history and clinical presentation and that "there was a temporal time course where she had a clear onset of symptoms, a complex of symptoms following the vaccine, within that temporal time frame, it was helpful in terms of thinking about what the ultimate cause may be for her symptoms." Id. He also testified that the appearance of enhancement on petitioner's MRI and EGM studies post-vaccination is consistent with his opinion that there was an acute inflammatory reaction occurring after the February 21, 2007 Gardasil vaccination. Tr. at 87, 92. Dr. Schechter also testified that there was no particular article or case study that led him to his opinion on causation in this case, but rather "there is evidence in the literature of… post-vaccine induced GBS, including Gardasil." Tr. at 60. In addition, Dr. Schechter testified that petitioner's clinical course and symptoms could not be explained by any other mechanism. Tr. at 39.

Dr. Axelrod, when asked by the undersigned how, by the concept of molecular mimicry, the vaccine could cause actual damage to the myelin, Dr. Axelrod explained that the body's immune response would recognize antigens that are homologous and then attack the body's myelin through the antigens or antibodies. Dr. Axelrod testified that it was the time interval between the vaccination and the development of petitioner's symptoms that further led him to his opinion that the vaccination was more likely than not the cause of petitioner's symptoms. Tr. at 149-50. Regarding any other explanation of how the vaccine could have caused damage to petitioner's myelin, Dr. Axelrod stated that he would have to defer to the expertise of a

neurologist, but he explained that petitioner's treating physicians looked for other possible causes of her symptoms and were unable to find one. Id. at 155.

Because Dr. Schechter was one of petitioner's treating physicians, the undersigned has given Dr. Schechter's opinion careful consideration. Capizzano, 440 F.3d at 1326 (treating physicians "are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.' ") (quoting Althen, 418 F.3d at 1279. The undersigned finds persuasive the opinions of petitioner's experts, Dr. Schechter and Dr. Axelrod, that the Gardasil vaccine caused petitioner to develop the Miller-Fisher variant of GBS, and finds that petitioner has satisfied her burden under Althen Prong Two.

(3) Prong Three: Is there a Medically-Acceptable Temporal Relationship?

The third Althen prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." Id. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." de Bazan v. Sec'y of Health & Human Servs., 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (Althen prong one's requirement). Id.; Koehn v. Sec'y of Health & Human Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro v. Sec'y of Health & Human Servs., 101 Fed. Cl. 532, 542 (Fed. Cl. 2011), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 2013 WL 1896173 (Fed. Cir. 2013).

Petitioner testified that the onset of her symptoms began on April 1, 2007 (39 days after her February 21, 2007 Gardasil vaccination), and she was admitted to WBH on April 4, 2007 (42 days after vaccination). Tr. at 9; Pet. Ex. 6 at 1-2. Dr. Axelrod and Dr. Schechter testified and state in their respective expert reports that an appropriate temporal association between vaccination and injury can be anywhere from one to six weeks. Dr. Schechter opined that the onset of petitioner's injury occurred during an appropriate time period, "that being between 1 week to 6 weeks." Pet. Ex. 57 at 1. Dr. Axelrod opined that GBS "may occur up to at least 6 weeks following vaccination with Gardasil." Pet. Ex. 23 at 2. Dr. Leist does not dispute that a vaccine reaction can occur as far as six weeks after vaccination. Thus, the undersigned finds that petitioner has satisfied Althen Prong Three.

e) Alternative Causation

Because petitioner has established a prima facie case, she is entitled to compensation unless respondent can put forth preponderant evidence "that [her] injury was in fact caused by factors unrelated to the vaccine." Whitecotton v. Sec'y of Health & Human Servs., 17 F.3d 374, 376 (Fed. Cir. 1994), rev'd on other grounds sub nom., Shalala v. Whitecotton, 514 U.S. 268 (1995); see also Walther v. Sec'y of Health & Human Servs., 485 F.3d 1146, 1151 (Fed. Cir. 2007).

In his report, Dr. Leist opined that the symptoms and the process that led to petitioner's facial weakness after her February 2007 Gardasil vaccine was the same process that led to her facial weakness prior to the vaccination. Tr. at 170. He also hypothesized that petitioner may have had a foodborne illness shortly after the February 2007 vaccination, which may have contributed to the increase of symptoms shortly after vaccination. Id. In addition, in respondent's prehearing memorandum, respondent argues that at least three of petitioner's treating neurologists (Drs. Muttal, Ernstoff, and Gruis) felt that petitioner's symptoms were not due to GBS or Miller-Fisher variant GBS, but were more likely due to a viral neuritis or undiagnosed connective tissue disease, and those neurologists did not attribute causation to the HPV vaccination. Resp't Prehearing Memo at 24.

As discussed above, a preponderance of the evidence establishes that petitioner's symptoms were of the Miller-Fisher variant of GBS, not symptoms of her pre-existing health issues or a food-borne illness as Dr. Leist suggests. In addition, while several of petitioner's treating physicians speculated about alternative causes of petitioner's condition, none of the physicians settled on any one cause as a more likely diagnosis, as was done for the diagnosis of the Miller-Fisher variant of GBS. At least five of petitioner's treating physicians either considered a diagnosis of the Miller-Fisher variant of GBS or actually diagnosed petitioner with that condition, while there was no consensus as to the other possible causes. Accordingly, respondent has failed to provide preponderant evidence of an alternative cause of petitioner's Miller-Fisher variant of GBS.

f) Significant Aggravation Claim

A significant aggravation is defined as "any change for the worse in a preexisting condition which results in markedly greater disability, pain, or illness accompanied by a serious deterioration in health." 42 U.S.C. § 300aa–33(4). As confirmed in W.C., 704 F.3d at 1357, the elements of an off-Table significant aggravation case were stated in Loving. There, the Court blended the test from Althen, 418 F.3d at 1279, which defines off-Table causation cases, with a test from Whitecotton, 81 F.3d at 1107, which involves on-Table significant aggravation cases. Thus, to prevail under a significant aggravation theory, petitioner must establish: "(1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a "significant aggravation" of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation." Loving, 86 Fed. Cl. at 144.

In her post-hearing brief, petitioner added a claim of significant aggravation claiming that the Gardasil vaccine caused a "substantial aggravation/exacerbation of an underlying demyelinating disorder." Petitioner's Post-Hearing Brief, filed Dec. 16, 2015 ("Pet. PH Brief"). In support of this claim, petitioner simply stated that: (1) "[a]n increased incidence of GBS has been observed after administration of the Gardasil vaccine," (2) petitioner "suffered GBS" and that the "onset occurred within the right time frame and her doctors have no explanation," and (3) the "onset of petitioner's illness occurred within the time frame after vaccination which is expected when an illness or vaccination causes GBS." Pet. PH Brief 2-3. Respondent filed a

post-hearing brief addressing petitioner's significant aggravation claim on December 23, 2014. Respondent's Post-Hearing Brief, filed Dec. 23, 2014 ("Resp't PH Brief"). Respondent disagrees that petitioner had a new post-vaccination demyelinating event. Resp't PH Brief at 5. Respondent argued that based on the record as a whole, petitioner failed to present preponderant evidence that she suffered from GBS, the Miller-Fisher variant of GBS, a de novo demyelinating injury, or a significant aggravation of an underlying demyelinating injury after vaccination. Id. at 7.

Because the undersigned has found that petitioner suffered a new event after vaccination, i.e., a development of the Miller-Fisher variant of GBS, and has also found that petitioner presented preponderant evidence to demonstrate the Gardasil vaccine caused her to develop this condition, there is no need for an analysis of petitioner's significant aggravation claim. Her causation-in-fact claim has succeeded.

VI. Conclusion

For the reasons discussed above, the undersigned finds that petitioner is entitled to compensation because she has provided sufficient circumstantial evidence that preponderates in her favor. A separate damages order will issue.

IT IS SO ORDERED.

s/ Nora Beth Dorsey
Nora Beth Dorsey
Special Master